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AN INITIAL REPORT FROM THE NEUROLOGICAL LABORATORY OF THE PHILADELPHIA POLYCLINIC.

UNDER THE DIRECTION OF

ALOYSIUS O. J. KELLY, A.M., M.D.

PATHOLOGIST AND CLINICAL ASSISTANT TO THE NEUROLOGICAL DEPARTMENT.

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UNDER THE DIRECTION OF

ALOYSIUS O. J. KELLY, A.M., M.D.,

Pathologist and Clinical Assistant to the Neurological Department.

The Neurological Laboratory is a part of the Department of Diseases of the Mind and Nervous System in charge of Professors CHARLES K. MILLS and CHARLES W. BURR, who furnish most of the specimens.

LESIONS OF THE BRAIN FOUND IN A CASE OF ACUTE YELLOW ATROPHY OF THE LIVER.

BY CHARLES W. BURR, M.D. AND ALOYSIUS O. J. KELLY, M.D.

THE clinical history of the case is as follows: B. D., male, aged forty years. His previous health had always been good. Having felt poorly for about one month, one week prior to his death he began to complain of chilliness, sweats and vague gastro-intestinal symptoms. His wife then noticed that he was slightly jaundiced. The jaundice rapidly increased in intensity, until before his death it was extreme. On the second

[1] Presented at the Joint Meeting of the Pathological Society of Philadelphia and the Philadelphia Neurological Society, held in Philadelphia, May 28, 1896.

day of his illness he began to be delirious and to have general convulsions. The liver dulness became very much decreased in area. The patient vomited often, and his bowels were absolutely constipated. The urine contained leucin and tyrosin, but no albumin. Two days before his death, his delirium passed into stupor, stupor into coma, and in coma he died, Dec. 28, 1895.

The clinical diagnosis was acute yellow atrophy of the liver.

At the necropsy the liver was about one-third the normal size, brown in color, and with wrinkled capsule. It was peculiarly soft to the touch and cut like india rubber. On section the liver structure was indistinctly visible in the right lobe, but absolutely abolished in the left. There was some oozing of bile. The gall bladder contained about an ounce of thick, greenish black bile, with a little bile sand. There was no obstruction of the ducts, no calculi. The stomach contained a few ounces of bile-stained fluid, but was otherwise normal. The spleen was slightly enlarged and soft. The kidneys were congested, the capsule stripped well; the cortices were well marked off. The brain macroscopically ap peared normal.

For the specimens we are much indebted to Dr. L. C. Peter.

The lesions discovered by microscopical examination of the brain may be divided into those found in sections prepared according to the Golgi method and its modifications, and those found in sections stained according to the Nissl methods and modifications. It is convenient to describe the latter first.

Sections from the motor and præfrontal regions of the cerebral cortex after the usual procedures subsequent to hardening in alcohol, were stained with dahlia, vesuvin, magenta red, methylene blue and thionin. While the majority of the stainings with the first mentioned agents were very satisfactory, none of them approached in clearness and sharpness of detail those stained with thionin, according to the method of Lenhossek. The stainings were far superior to those obtained with the much vaunted methylene blue, which latter were, however, better than those obtained with the other dyes. What follows, therefore, refers particularly to thionin preparations, though precisely similar changes were disclosed by the other stainings.

The pathological lesions discovered affect the nucleolus, the nucleus and the cell body (Fig. 1). Normally,

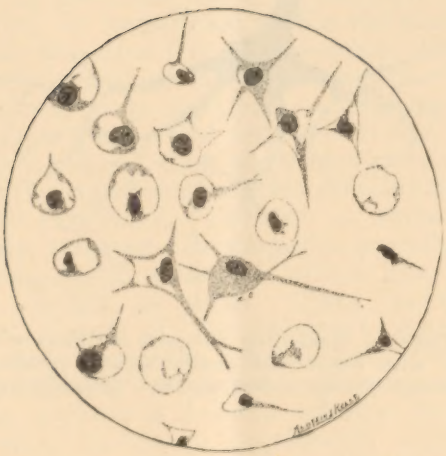

FIG. 1. Drawing illustrating parenchymatous degeneration of the cells of the cerebral cortex; implication of the nucleolus, nucleus, and cell body. The cells in the upper half as in a microscopic field; in the lower half, collected from various regions. N. C., normal cell for comparison. Thionin preparation. x 1200 Zeiss.

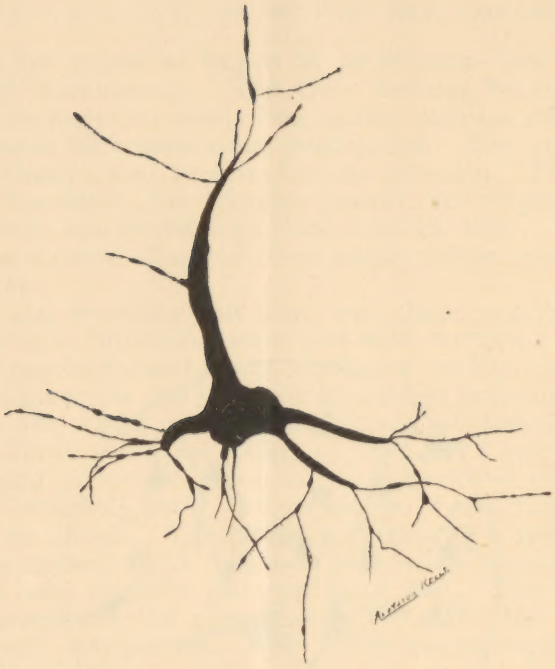

FIG. 2. Drawing illustrating irregular swellings or tumefactions of the dendrites, with loss of the gemmulæ, of a pyramidal cell of the cerebral cortex—early stage of degeneration. Golgi-Berkely preparation.

FIG. 3. Drawing illustrating atrophy and disappearance of the dendrites—late stage of degeneration. Golgi-Berkely preparation.

the nucleolus is of a definite size, of rounded contour, with edges smooth and well defined, and has a certain marked, not very variable affinity for the stain. In our preparations the nucleolus is very slightly, if at all, increased in size; it is of somewhat irregular contour, which irregularity is not marked, consisting when demonstrable of a slight and limited projection into the substance of the nucleus at one, occasionally two points, along its periphery; its edges, while sharp and well defined, are not as smooth as normally, and its receptivity of the stain is markedly increased, it appearing as a jet black dot, rendering absolutely futile all attempts to recognize any structure. In its position within the nucleus we can recognize no deviation from the normal, it being situated here and there irregularly as normally.

The nucleus, while apparently exhibiting no marked deviations from the normal variation in size, is frequently distorted in shape, being often elongated, again quite irregular with projections and indentations along various parts of its periphery. The two or three larger and more deeply staining chromophilic particles normally present irregularly arranged within the nuclei, are no longer visible as such. They have lost their identity by reason of the excessive staining which the other finer dust-like chromophilic particles have thus pathologically taken on. The normally clear karyoplasm evinces a decided affinity for the stain, being in many instances excessively stained. This staining of the normally clear karyoplasm and the excessive staining of the nuclear chromophilic particles in many instances, produces such an intense staining of the nucleus as a whole as to obscure the nucleolus, which, in other instances can be quite distinctly recognized.

The cell body seems to be most intensely affected, their being present all gradations from slight implication to almost absolute destruction of it. The chromophilic particles, which normally are distributed throughout the cell body with a certain regularity usually in rows, are in our preparations very irregularly arranged, often showing very curious figures. We notice in some cells but a slight deviation from the normal, the chromophilic particles having disappeared from but a small area, and being possibly more densely aggregated in another region. From this slight change there are visible all gradations of destruction until there remains but the nucleus deeply stained, surrounded by a few particles still stained along the periphery of the cell

body. In many instances the chromophilic particles are excessively large. One is inclined to think of a coalescence of smaller ones, as high magnifications (1200) fail to reveal any distinction into parts. In cells manifesting all grades of destructive changes, there is usually arranged around the nucleus a layer of fine, stained dust-like particles, between which and the nucleus, there is always a clear unstained zone.

In some instances the nucleus and cell contents have been entirely destroyed, or the nucleus has dropped out of the cell, and all that is discernable are a few fine stained particles along what was the periphery of the cell. In many cells which are not so excessively affected by the destructive process, there is an appearance extremely suggestive of fat globules and detritus. The fat therefrom may unfortunately have been removed during the process of hardening.

The protoplasmic processes as far as they are stained present in some instances changes similar to those described as affecting the cell body. In most instances, however, they exhibit no deviation from the normal.

The pathological changes described affect all varieties of cortical cells. We have been unable to discover that any one variety is more affected than another. In spots, however, the destructive process has been more marked than elsewhere. In certain regions all the cells in a quite considerable area are more or less destroyed, while in others but few cells are implicated. This applies to all the layers of cortical cells. Again, in a few places it seems as though the more superficial cells were more intensely affected than those of the deeper layers. These instances, however, are very few.

Other pieces of cortex after hardening in Müller's fluid were stained according to the silver phosphomolybdate method of Berkely. The difficulty of determining, by this and the ordinary Golgi method of staining, the number of cells affected is very great owing to the uncertainty of the impregnation and to the fact that of all the cells of the various layers of the cortex but a very few are at any one time stained. This latter fact is, however, a great advantage, as we are thereby enabled to study much more readily the changes affecting the individual cells and their prolongations. This method of staining is inappropriate for detecting the finer changes in the cell body, and in our preparations gave negative results. The changes affecting the prolongations are, however, quite definite. They are of two kinds, or, as we

prefer to say, two stages of one process. The first consists of a swelling (Fig. 2), the second of an atrophy of the processes (Fig. 3).

The first are more numerous. They consist of irregular swellings or tumefactions which extend along the dendrites for a variable distance, and are of variable thickness. Some are very minute, others quite large. The apical process itself is very frequently affected close to the cell body proper. In other instances the parent dendrite and its finer branches abruptly become thickened, and as abruptly resume their normal calibre. As a rule, throughout the course of these tumefactions the gemmulæ are absent, though occasionally one still detects a few projecting from the periphery of the swellings. Along some of the finer dendrites not the subject of such pathological tumefactions, the gemmulæ have also disappeared. In other instances they themselves appear somewhat swollen. The varicosities normally present at the point of branching of the dendrites appear in many instances to be excessively large.

The further stage of this destructive process has already been alluded to as an apparent atrophy. The gemmulæ have entirely disappeared from the dendrites—a very few may occasionally still persist. The dendrites themselves have become thinned and shortened. That they have not been broken or cut off during the process of preparation, is evident from the fact that at their distal extremities they appear rounded and well defined. Further, many of the dendrites have entirely disappeared. In some of these latter instances, as well as in some of the former, the apical process is still quite thick.

This destructive process affects apparently indiscriminately the various cells of the several layers of the cerebral cortex. The determination of the number of the implicated cells is, however, as before stated, impossible. We could detect no pathological changes in the axis-cylinders or collaterals.

The study of the cerebellum led to very indefinite results. Aside from variations in the intensity of the staining of the Purkinje cells, nothing of any note was discovered by the use of the aniline dyes. The impregnation of the cells of the cerebellum by the Berkely modification of the Golgi method was less satisfactory than was that of the cells of the cerebrum, but the alterations found in the Purkinje cells were practically the same as those described affecting the cells of the cerebral cortex. We detected no change in the neuroglia elements of

either the cerebrum or cerebellum, and no disease of the blood-vessel walls.

To sum up we have found evidences of a marked destructive process affecting the bodies and the processes of the nerve cells of the cerebral cortex. The lesion is parenchymatous affecting the nerve cells primarily, and is not secondary to any disease of the blood vessels. We are entirely ignorant of the primary cause of this destructive process. It is more than probable, it is almost certain, that acute yellow atrophy of the liver is caused by a poison, and that it is not a local but a general constitutional disease, a toxemia. And since cerebral symptoms, delirium, perhaps mania, general or local convulsions and coma form no small part of its clinical history, we should expect to find distinct signs of disease in the cortex whether such signs be the result of penetrated function or its cause. The fact that heretofore microscopic study of the brain in conditions of delirium, acute mania, and the acute infectious fevers has frequently been fruitless or indefinite, proves nothing, since we are only now beginning to learn methods of research competent to show any of the finer microscopic changes. As methods have improved, the number of so-called functional diseases has steadily decreased. Granting that acute yellow atrophy of the liver is a toxemia we are confronted with the question as to whether the brain lesions found are due to the same poison or whether they are due to another or to others produced by disturbance of any of the body functions. We cannot answer it. We have, of course, assumed that the lesions found by us in this one case are not accidental, but essential.

Whether this be so can only be proven by the study of other cases. The lesions found are, of course, not claimed to be characteristic of acute yellow atrophy of the liver. Indeed, we should expect to find similar changes in the acute infectious fevers accompanied by similar brain symptoms. As a matter of fact the lesions found in this case by using Berkely's modification of Golgi's method correspond to those found experimentally by Berkely in ricin and alcohol poisoning and are not unlike those described by Andriezen and others as occurring in certain types of insanity, notably alcoholic dementia.

That the case was one of acute yellow atrophy of the liver is proven not only by the clinical history and the necropsy, but also by the microscopical examination of the liver, spleen and kidneys.

SENILE PARAPLEGIA.

By ALOYSIUS O J. KELLY, M.D.

THE clinical history of the case is as follows: G. B., aged 79 years, widower, white, native of Scotland, jeweller by occupation, was admitted to the out-wards of the Philadelphia Hospital, May 3, 1895, and to the men's nervous wards of the same hospital, February 13, 1896, and the following notes obtained:

No history of any hereditary disease. He was treated in the surgical wards of the Philadelphia Hospital during September, 1895 for urethral stricture, and subsequently again transferred to the out-wards. He has been a moderate drinker; syphilis is denied. About three months prior to his admission to the nervous wards, he noticed that he was weak in the legs. He never had had any distinct apoplectiform attack. Two days before his admission, on attempting to arise in the morning he found that he was paralyzed from the waist down. He had retention of urine and incontinence of feces.

Examination on admission from the out wards revealed complete paralysis of all the muscles below the waist. There were no areas of anæsthesia. The third day after the paralysis came on, bed sores began to form over the sacrum and heels. Although he was put on a water bed, these bed sores rapidly became worse. Examination of the urine showed a trace of albumin and a large number of granular casts. Symptoms of uremia began to develop and spots of consolidation in both lungs could be detected. The fourth day after his admission he became delirious. The temperature never went above normal. He became rapidly weaker, and died seven days after admission, apparently of uremia. (February 20, 1896, 5 P.M.)

Post-mortem examination by Dr. Jamison, February 22, 1896, 11 A.M. Body of well-nourished old man, a small bed sore over the left heel and a large one over the sacrum. Bladder distended and inflamed. Kidneys show parenchymatous and interstitial nephritis. Heart nor-

mal. Lungs emphysematous; a few spots of senile pneumonia. No gross lesions of the brain or spinal cord, except a chronic leptomeningitis.

For the specimen (spinal cord) and the above notes, I am very much indebted to Dr. Charles K. Mills.

Microscopical examination of the cord revealed changes which, though intimately associated in a causal relationship, it will be convenient to describe under two headings, first, those affecting the nerve fibres; second, those affecting other tissues.

Upper cervical region: In the postero-median columns, close to and parallel with the posterior two thirds of the median fissure, there is a narrow area in which the nerve fibres are diminished in number and in thickness. There is a quite marked degeneration of conical shaped area with base inward, bounded by the anterior one third of the posterior fissure, the posterior commissure, and the posterior cornua, and extending pointedly into the postero-external columns almost to the periphery. In the lateral columns not strictly localized to the pyramidal tracts although there much more marked, the nerve fibres are degenerated in part.

Cervical enlargement: The degeneration in the postero-median columns along the posterior fissure is more marked than in the previous section, while the conical-shaped area alluded to, is larger. The degeneration in the lateral columns is marked, but is not limited to the pyramidal tracts While these tracts are much more affected than are other regions, the degeneration extends forwards into the anterior ground fibres.

Lower cervical: The degeneration in the posterior columns, while more marked along the posterior median fissure is distributed to some extent throughout the columns. The degeneration in the lateral columns as heretofore affects particularly and more markedly the pyramidal tracts, though it is not confined to them.

Upper, middle and lower thoracic regions: The degeneration in the posterior columns is much more general and much more marked than in previously described sections The degeneration in the lateral columns is of about the same extent as in the last mentioned section.

Lumbar cord: The degeneration in the posterior columns is very marked. In the lateral columns as heretofore.

Sacral: The fibres along the lateral margin appear fewer in number and narrower than elsewhere

FIG. 1. Drawing showing the excessive intra-spinal overgrowth of connective tissue and its sources of origin, the sclerosis of blood vessels and pia mater. Ammonio-carmine preparation.

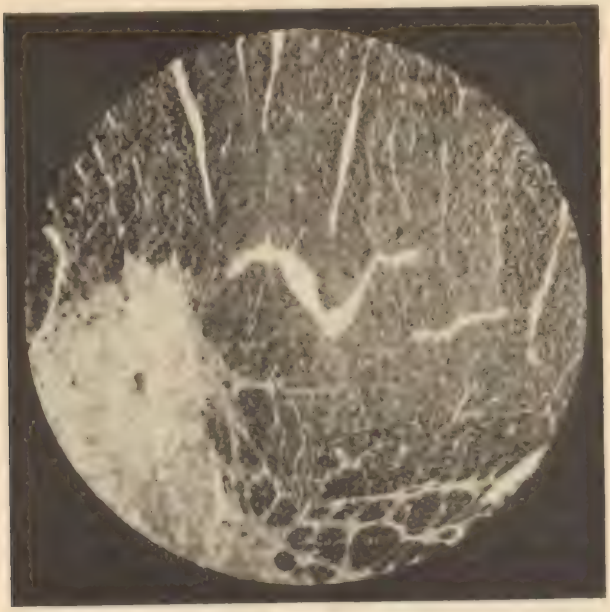

FIG. 2. Photograph showing the blood-vessel sclerosis, the associated perivascular connective tissue overgrowth, and the numerically increased and much thickened trabeculæ of connective tissue and the numerous islets of sclerosis in connection therewith. Weigert-Pal preparation.

FIG. 3. Higher magnification of Fig. 2.

FIG. 4. Photograph showing changes similar to those evident in Figs. 1., 2. and 3. Hæmatoxylin—Picric Acid—Fuchsin preparation.

When not otherwise stated, the nerve fibres of the various tracts appear normal.

Throughout the cord at all levels, but especially in the regions noted as revealing nerve fibre degeneration that is, in the posterior and lateral columns—there is a great increase of the connective tissue. This connective tissue overgrowth is intimately associated with changes in the blood vessels and pia mater. With the exception of the very minutest ones, the blood-vessels are everywhere distended, and all have greatly thickened walls. This applies to vessels of all calibres, from minute spinal ones to larger pial vessels. The smallest spinal vessels have greatly thickened walls and the lumen of many of them has apparently become obliterated. Careful examination of somewhat larger ones, reveal slight proliferation of the intima and great increase in the thickness of the media, which is poor in nuclei, concentrically laminated, and apparently somewhat hyaline in structure. Surrounding this latter is a layer of variable thickness of less homogeneously staining connective tissue. These changes are very marked in some of the median sized and larger vessels, which themselves are occasionally the seat of some round cell infiltration.

The pia-mater is very greatly thickened, contains a very few nuclei, and has become united with the arachnoid. The peripheral layer of the spinal neuroglia is also greatly thickened. From this greatly thickened pia and neuroglia, and from the blood-vessels with thickened walls as noted above, there extend throughout the cord numerous connective tissue trabeculæ. These are very greatly in excess in number and much thicker than normally, and while present throughout the cord, are much more in evidence in the areas already referred to as revealing nerve fibre degeneration.

These trabeculæ are frequently very thick, inclosing within their confines bundles of nerve fibres, whereas, again they are finer and surround only individual fibres, leading thus to degeneration of single fibres. The blood-vessel sclerosis is, of course, not confined to the vessels of the white matter of the cord, but implicates of necessity those supplying the gray matter, which latter is not, however, appreciably altered. The central canal is filled with proliferated epithelium.

The most evident and important pathological alteration discovered in these sections is, therefore, the excessive overgrowth of connective tissue; the nerve fibre

degeneration being very properly considered secondary thereto. The intimate association of this sclerosis with the blood-vessels is very patent. The blood-vessels themselves are the seat arteritis and endarteritis, and around them as foci proceed trabeculæ of connective tissue encircling in places numbers, in others, fewer nerve fibres. The neurolgia along the periphery of the cord is also greatly thickened, and from it proceed also into the cord, trabeculæ of fibrous tissue, much increased in thickness and in number.

This sclerosis originating around the vessels and periphery of the cord, while marked in all regions of the cord, is, however, much more intense in the posterior and lateral columns, especially in the area of the pyramidal tracts, and is more marked in the thoracic region than elsewhere. In areas slightly affected, more particularly where the sclerosing tissue surrounds groups of nerve fibres, rather than individual ones, the fibres for the most part appear normal. In other regions, however, in which the sclerosing connective tissue envelops frequently individual fibres, these have suffered greatly therefrom and have become atrophied, axis cylinder and medullary sheath in many instances, having disappeared, in others being much narrower than normally. There remain, therefore, in consequence, many minute islets of fibrous tissue. In places where several larger vessels are in close opposition there are quite large spots of sclerosis.

There was discovered no embolic, thrombotic, hemorrhagic, or other process to account particularly for the acute manifestation of the severe symptoms. The case is simply an illustration of one of the many instances with which the neuropathology abounds in which definite lesion are for a long time devoid of any very manifest symptoms. These when they do come on, frequently make their appearance abruptly.

That the patient, however, was not entirely without symptoms prior to the acute attack of paraplegia, is evidenced by the weakness of his legs. And, in the light of the microscopical examination, and reasoning *a priori*, one may very safely assume that there were present other symptoms, unfortunately overlooked as is but too frequently the case in the gradual and progressive weakness which attends old age. One cannot, therefore, but regret imperfect history.

The designation "Senile Paraplegia," is admittedly

ill-chosen, but has been selected because of its conciseness, and because it is sufficiently expressive of the clinical condition. We do not, however, desire to be understood as suggesting that the changes described are essentially senile in nature. They are distinctly pathological, and while frequently an accompaniment of old age, they are to be considered apart from changes of a purely senile character. It will hardly be appropriate to here enter upon the discussion of this subject in detail. For the photographs I am much indebted to Dr. Schively.

THE SPINAL CORD LESIONS IN A CASE OF FRACTURE OF THORACIC AND LUMBAR VERTEBRÆ.

By ALOYSIUS O. J. KELLY, M.D.

THE clinical history of the case is as follows: W. T. A., male, aged 33 years, mason by occupation, was admitted to the Orthopædic Hospital under the charge of Dr. Wharton Sinkler, March 28, 1895. He had had gonorrhœa when he was 25 years of age, but syphilis was denied. December 30, 1891, a scaffold on which the patient was working, gave way, causing him to fall twenty feet to the ground. He struck on his heels, then on his back. A brick standing on end struck him in the lumbar region to the right of his spinal column, causing a bruise which persisted for some time. On attempting to arise he could not move his legs. He began to immediately experience a sensation as of "pins and needles" from his waist down. For four days after the accident his legs were painful and felt cold. He had complete loss of control of his bladder and rectum. His sexual function was abolished.

Examination on admission to the hospital: No eruption or œdema; deep bed sores over the sacrum, on each heel, and on the calf of the left leg. No movements in the legs, no rigidity. Loss of control of the bladder and rectum. Complete anæsthesia below the crests of the ilea; touch, pain and temperature sense all abolished. No girdle pain. No tenderness on pressure over the nerve trunks. Elbow jerks equal on both sides; muscle jerks in arms normal. Knee jerks absent, not reinforcible. Tendon achilles jerks absent; plantar jerks absent; cremasteric jerks present but much retarded. Abdominal reflexes present. Urine contains a trace of albumin.

Electrical examination (Dr. Willets): No reaction to the faradic current in any of the muscles of the legs. Galv. ancle. > Kelc. 35 ma. required to secure reaction in tibialis anticus; calf muscles 30 ma. Galvanic qualita

FIG. 1. One cm. above lesion. Photograph showing distortion of the cord, and complete degeneration of all the nerve fibres, except a few of those of the anterior column. Weigert-Pal preparation.

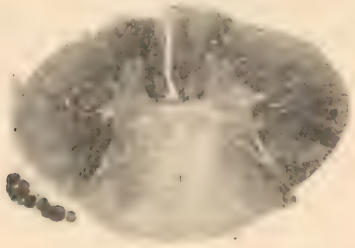

FIG. 2. Thoracic Cord, three cm. above lesion. Photograph showing degeneration of the nerve fibres of the posterior column and of those along the antero-lateral periphery (direct cerebellar and antero-lateral ascending tracts). Weigert-Pal preparation.

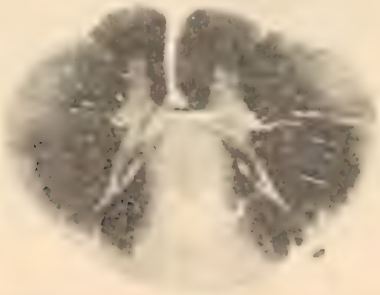

FIG. 3. Thoracic cord, nine cm. above lesion. Photograph showing degeneration of the postero-median, postero-external (partial), direct cerebellar and antero-lateral ascending tracts. Weigert-Pal preparation.

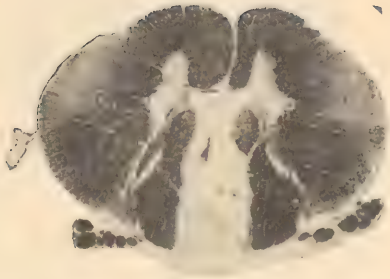

FIG. 4. Lower cervical cord, sixteen cm. above lesion. Photograph showing degeneration of the postero-median, direct cerebellar and antero-lateral ascending tracts. Weigert-Pal preparation.

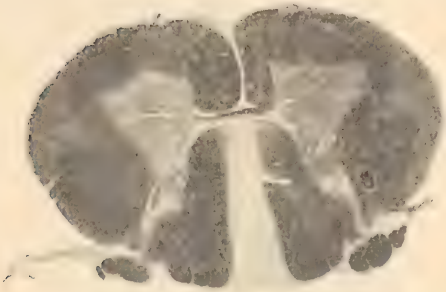

FIG. 5. Cervical enlargement, twenty-two cm. above lesion. Photograph showing degeneration of the postero-median, direct cerebellar and antero-lateral ascending tracts. Weigert-Pal preparation.

tive and quantitative change. Pupils equal and react well to accommodation and to light.

October 7, 1892, the patient died. The autopsy revealed a fracture dislocation of the first lumbar on the last dorsal vertebra; the dura mater adherent to the bodies of the vertebræ at the seat of fracture; the entire lumbar cord and the lower dorsal cord much flattened; chronic cystitis; slight dilatation of the ureters; pyelitis; abscesses in the kidneys.

For the specimen (the spinal cord) and the above notes, I am very much indebted to Dr. Charles W. Burr.

Microscopical examination (Weigert-Pal preparations): A section through the centre of the area of disease reveals total destruction of the cord, it being impossible to recognize any of its various constituents. What was the cord is now a mass of fibrous connective tissue, through which run a few partially degenerated nerve fibres. The nerve fibres of the cauda equina, particularly the most external ones, still retain their myeline sheath. Surrounding the structures of this region is a thick mass of dense fibrous tissue.

A section through the cord one centimetre above the former, shows great distortion of the cord, particularly of the postero-lateral regions, the component parts of which cannot be separately recognized. The cord is flattened from before backward, the posterior cornua being apparently displaced laterally, the posterior and lateral columns being crushed antero-posteriorly. The normal configuration of the anterior cornua and columns is well retained. There is complete degeneration of all the nerve fibres in this region of the cord, with the exception of a few along either side of the anterior median fissure, a few of the most centrally located of the anterior columns, a few skirting the medial and anterior border of the anterior cornua, and a few of the anterior commissure (Fig. 1).

A section through the cord 3 cm. above the first, shows the cord to be about normal configuration. Along the antero-lateral periphery there is a degeneration of the nerve fibres of varying extent, being greatest just a little external to the posterior cornua. With the exception of a few fibres bordering the tips of the posterior cornua, the fibres of the posterior columns are degenera ted in their entirety (Fig. 2).

A section through the cord 9 cm. above the first reveals a complete degeneration of the fibres of the postero-

median (Goll's) column, and some degeneration of the medial peripheral fibres of the postero-external (Burdach's) column. There is some degeneration of the fibres along the periphery of the cord between the anterior and posterior cornua of both sides. This degeneration is most marked about midway between the cornua, at which place it penetrates some distance into the substance of the cord (Fig. 3).

A section (lower cervical) 16 cm. above the first, shows complete degeneration of the posterior median columns, which degeneration towards the posterior commissure spreads out fan-shaped. The postero-external columns are otherwise unaffected. The degeneration along the lateral periphery noted in the previous section is more marked, and about midway between the anterior and posterior cornua extends quite a considerable distance into the substance of the cord (Fig. 4).

A section (cervical enlargement) 22 cm. above the first reveals the same general characteristics as those evident in the last described section (Fig. 5).

Below the seat of manifest lesion—in the conus—it is absolutely impossible to recognize any spinal cord structure. There is present a mass of connective tissue through which run a few partially degenerated nerve fibres.

When not otherwise mentioned the tracts are normal.

There is, therefore, complete disorganization of the cord at the seat of fracture, and above this region throughout the cord, ascending degeneration of the postero median, direct cerebellar, and antero-lateral ascending (Gower's) tracts; and for a short distance, degeneration of the postero-external columns. In other words, we find the anticipated spinal cord lesions of a fracture in this region. To be mentioned only is the degeneration of the direct cerebellar tract in conjunction with a fracture of the vertebræ so low down.

For the photographs, I am very much indebted to Dr. Schively.

A CASE OF PRIMARY COMBINED COLUMN DISEASE.

By JOHN H. W. RHEIN, M.D.,

Instructor in Nervous Diseases, Philadelphia Polyclinic.

MRS. L., aged 59 years, presented herself for examination, complaining of unsteadiness in walking and numbness from the waist downward. Her family history reveals a decided neurotic taint. Her grandmother and three aunts had paralysis agitans, a sister nystagmus, and her mother an attack of apoplexy. She herself had enjoyed excellent health prior to the onset of the present affection, which began, she stated, suddenly, about two years previously.

There was an attack of unconsciousness preceded by stiffness in the neck and nausea. No paralysis followed, but shortly afterwards distinct insecurity in walking, with progressive weakness in both legs developed. She became emaciated and extremely pallid.

When examined by Dr. Frances Janney, eighteen months after the onset, she presented the following symptoms:

The patient was exceedingly anæmic, and moderately emaciated. There was no paralysis, though the legs were weak. The wasting was general and did not suggest atrophy from central trouble. The station was poor with feet together and eyes open or closed. The gait was distinctly ataxic. The knee jerks were decreased on both sides, but reinforcible. The plantar reflex was retained. There was no change in sensation except paræsthesia of the legs and trunk to the waist. A vaginal examination revealed the presence of a large tumor involving the fundus of the uterus. Its existence had hitherto not been suspected.

Some months later a second examination was made and the above condition confirmed. The knee jerks were irregular. On the right the response was slightly below normal, on the left almost normal, on both sides reinforc-

ible. There was no clonus or ankle jerk. The elbow jerk and chin jerk were present. Muscle reaction was good. Pain sense and sensation to touch and heat were normal. The paræsthesia was still present.

There was no disturbance of the bladder or rectal functions. A fine rythmical tremor in both hands was observed. This had existed for years and was not made worse by voluntary effort. An examination of the blood made by Dr. James E. Talley, showed 2,464,000 red blood cells and 40% hæmoglobin. Dr. A. G. Thomson reported the eye condition as follows: Pupillary reaction normal, form fields and fundi normal. Clear media, margins of discs hazy. The patient died a year later from exhaustion. Her exact nervous condition at the time was not studied.

The necropsy revealed the following facts: In the uterus, involving the whole body of this organ, was a large, hard tumor, which microscopic section proved to be a fibro-sarcoma. Some few masses, small in size and scattered, were seen in the liver, which was normal in size. The examination otherwise proved negative, except the spinal cord, which was the seat of a lesion presenting the greatest interest. Microscopically the cord appeared smaller than normal. There was no meningitis. The brain and its enveloping membranes showed no change. Unfortunately, the brain in the process of hardening, underwent decomposition, hence a report of the microscopical condition of this organ is impossible. Macroscopically there was demonstrated degeneration in the posterior and lateral columns of the spinal cord, as seen by the distinct paling of these tracts. The process involved the whole extent of the cord and was most intense in the lower dorsal region. The degeneration in the lateral tracts was not so extensive as in the posterior columns at any of the levels.

The cord was hardened in Müller's fluid, imbedded in celloidin, and stained by the Weigert-Pal method and with ammonio-carmine.

Microscopical.—After staining, a sclerotic change was seen in the posterior and lateral pyramidal tracts throughout the whole extent of the cord, almost systemic in character. In the posterior columns the degeneration is most intense in the dorsal region, least in the lumbar region, while in the cervical region it is considerably more than moderate. There is always a strip of healthy tissue surrounding the degenerated area in the posterior columns

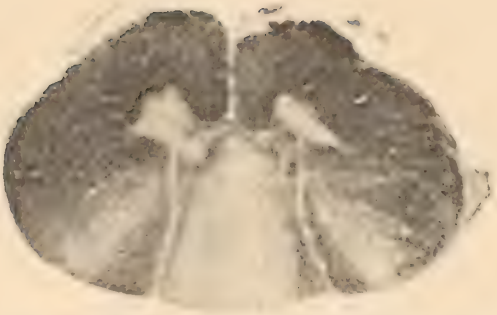

FIG. 1. Cervical cord showing degeneration of the postero-median, the postero-external (in part), and the crossed pyramidal tracts.

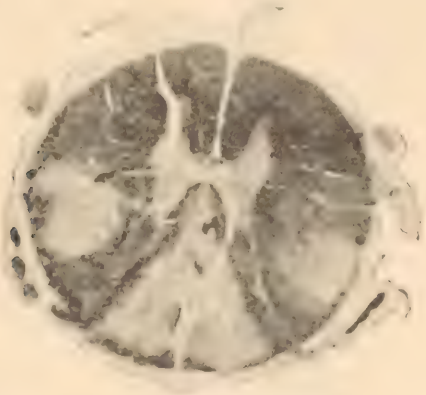

FIG. 2. Thoracic cord showing degeneration in the posterior and lateral columns.

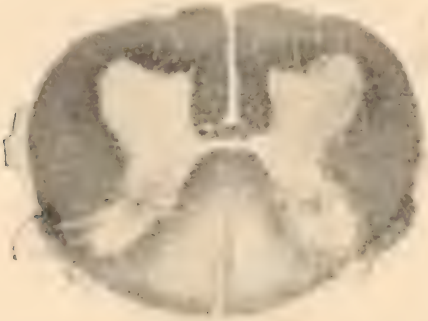

FIG. 3. Lumbar cord showing degeneration in the posterior columns.

and the columns of Goll are more intensely involved than the columns of Burdach.

The ganglion cells in the anterior horns show slight change in places, chiefly in the upper dorsal region. The posterior roots and Clark's column show no signs of involvement.

In the upper cervical region, the degenerated area involves the columns of Goll in their entirety, except a few fibres medianward and cervically, which take the stain well. Though to a much less degree, the columns of Burdach are markedly involved. Centrally there remain a number of healthy fibres, while peripherally but a few well-stained fibres can be seen. The root zones are present. In the lateral pyramidal tracts there is beginning degeneration, but to a much less degree than is seen in the posterior tracts. The anterior pyramidal and cerebellar tracts are intact. The anterior horns and posterior roots as well as the remaining tracts of the cord are normal.

In the cervical development, the same condition exists save that the degeneration in the postero-external columns and in the lateral pyramidal tracts is more intense.

In the dorsal region the process has so far progressed that few healthy fibres remain in Goll's columns. In the columns of Burdack a narrow strip along the posterior horns alone is stained, the process growing more intense from above downwards. In the anterior pyramidal tracts a very moderate degree of degeneration is observed. There is marked degeneration of the crossed pyramidal tract. In the upper levels a few ganglion cells have lost their prolongation, but there seems to be no lessening in the number. The process involved to a very slight degree the antero-lateral ascending tracts.

In the lumbar region the degeneration is less marked, especially marginally. In the posterior columns healthy fibres in increasing numbers are observed. A band of well stained tissue encircles completely the degenerated area.

In the lumbar enlargement, only traces of the process in the lateral columns are seen. In the posterior columns are two wedge-shaped areas of degeneration on either side of the posterior fissure, surrounded by healthy tissue.

The degeneration now rapidly becomes less in extent and degree, until in the sacral cord there is no trace of

the process visible. Clark's columns and the posterior roots are intact.

The carmine specimens show smaller axis-cylinders; in places many entirely disappeared; a marked increase in connective tissue. To a very moderate degree is there increase in the blood vessels. There were seen no granular cells in any of the sections.

This is evidently one of a group of cases which has been variously described as combined tabes, postero-lateral sclerosis, spastic tabes (Grasset), ataxo-paraplegic tabes (Déjerine), ataxic paraplegia (Gowers), combined fascicular disease, progressive spastic ataxia (Dana), true combined system disease (Babinski and Charrin) and primary combined column disease (Rothman).

In 1871 Prevost described the post-mortem findings in what was probably the first case belonging to this group. Later, Babesin, Strümpell, Siali, Westphal and others added cases to the literature of the subject. In 1886 Grasset collected 33 cases with autopsies and called the disease combined tabes (ataxo-spasmodique). The evidence did not point to a mere systemic lesion. He believed with Gowers that while the lesion in the posterior columns was systemic, in the lateral columns it tended to be diffuse, and concluded that the process was a diffuse myelitis.

A study of the autopsies in the cases above demonstrates clearly that the disease is one with a most varied morbid anatomy, different columns in varying degrees of intensity being involved. The gray matter may or may not be diseased; the peripheral nerves have been found involved; Clark's columns and the posterior roots as shown by Rothman, Mayer and others are often affected. On the other hand, a number of cases show a noteworthy unanimity in the distribution of the lesion in the posterior columns. With moderate constancy the lesion is more intense in the lower dorsal region, less in the cervical region and least in the lumbar and sacral regions. As a rule there is a band of healthy fibres surrounding the diseased areas adjacent to the posterior horns. In the lateral columns the degeneration is not strictly systemic as Gowers, Dana and others have proven, and which is shown in the case under discussion.

In some cases the lateral mixed columns are encroached upon, and the direct cerebellar tracts are not infrequently involved. In Strümpell's case, reported in 1880, there was systemic degeneration of the pyramidal

tracts, posterior columns, cerebellar tracts with atrophy of Clark's columns. The anterior horns and peripheral nerves escaped.

This variability in the morbid anatomy, together with the irregularity in the clinical history, suggests that the lesion is not truly systemic, but rather diffuse, beginning as a primary disease of the column of the cord, the posterior and lateral pyramidal tracts most usually, and developing as the process progresses into a diffuse one. It is not a systemic lesion, as Strümpell, Hochhaus and others maintained. It may, if the patient succumbs early, show a limitation to certain tracts in the cord, but when the disease is of long duration the process takes on an increasingly diffuse character. Strümpell believed that the further extension of the disease by continuity is improbable. Certainly, at first the limitation to certain columns, and the freedom of the parts immediately adjoining the diseased areas, inclines to this view, but later there is little doubt that continuity plays at least some part in the extension of the lesion.

The presence in the case under discussion, of a malignant tumor of the uterus is interesting in connection with the recent reports of spinal degeneration in constitutions with morbid blood states. Lichtheim, Nonne, Minnich, Burr and others have described characteristic lesions of the cord in pernicious anæmia. Babes and Kalindero, in 1890, reported a case of Addison's disease, with lesions in the posterior and lateral columns with a distribution which suggests to no slight degree that found in many of these cases of combined sclerosis. Fleiner quotes Abegg's case of spinal degeneration in a case of Addison's disease. There was change in the posterior columns in two of his own cases.

Minnich mentions spinal change in three cases of chronic icterus, in one case of leukæmia, in a case that died of tumor of the "vermis inf. cerebri," in two cases of chronic nephritis, and finally, a case of carcinomatous cachexia with hydræmia. Lastly, spinal lesion has been found in hypnotismus, pellagra, lathyrismus, and lead and alcohol poisoning. This clearly demonstrates some relation between toxic blood conditions and spinal degeneration, but just what that relation is, it is impossible in the present light of our knowledge to decide. In pernicious anæmia, the origin of the blood state remains as obscure as the cause of the spinal lesion. The theory that the two conditions are common results of the same

cause, probably a toxine seems the most plausable explanation, one is justified at present in making.

Since the exact duration of the tumor of the uterus is not known, we are inclined to place no importance upon its presence, and all the more likely to consider its presence a coincidence.

In studying the etiology of the affection it will be seen that nervous heredity plays an unimportant part; that syphilis is rarely an antecedent; that it is more frequent in males; that it is a disease of adult life; that exposure and excesses, exercise, predispose, and lastly, it follows spinal injury according to Rothman. The symptoms are characteristics. The onset is gradual, the first symptom being unsteadiness in walking. Soon there is added stiffness in the muscles, though this may be absent. Romberg's symptom is present and the gait is distinctly ataxic, though differing from the walk of true tabes. The high raising and flopping down of the feet is not prominent. It is a mingling of the gaits of spastic paralysis and true tabes—when spastic symptoms are present. Lighting pains are almost always absent owing probably to the freedom of the root zones. Other sensory symptoms are rare. The reflexes show a wide difference from the tabes. The knee jerks are usually much exaggerated or spastic at first, while towards the last they may be almost or quite absent. The plantar reflex is present, as well as the remainder of the superficial reflexes. The sexual power is early lost. The eye symptoms sometimes, but rather exceptionally, resemble those found in true tabes. The iris reflex to light is usually preserved and optic atrophy is the exception. Progressive weakness ushers in the final symptoms.

The disease has little tendency in itself to cause death according to Gowers and Dana, and usually is of long duration. Rothman's experience, however, leads him to conclude that the duration of the disease is but three years. In our own case the patient expired just three years after the appearance of the first symptoms, but the case was not uncomplicated, and had the malignant tumor been absent we are warranted in supposing that the patient would have survived a longer period.

In conclusion, thanks are due Dr. Frances Janney for her kindness in permitting this report and for the opportunity of making the necropsy. To the kindness of Dr. Schively are we indebted for the photographs.

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SPINAL CORD FROM A CASE OF POTTS' DISEASE.

By HENRY D. BOYER, M.D.

THE following case was under the care of Dr. J. P. Willits in the Germantown Hospital in the spring of 1894.

Unfortunately he was an Italian who spoke no language other than his own, and his previous history could not be ascertained.

When brought to the hospital he was a middle-aged man, with the objective symptoms of a chronic myelitis. And no direct cause for the cord disease could be found. He had evidently been in bed for some time. There were much emaciation, complete paraplegia, and contractures of the thighs and the legs with fibrous anchylosis at the hips and the knees. The sensation was completely lost from the umbilicus down. The knee jerks could not be tested on account of the contractures. There was full control over the bladder and the bowel sphincters. The arms were in no way affected, except by the general weakness. The back was perfectly straight. There was no point of especial tenderness along the spine, nor any pain in any other part of the body. The temperature was at times hectic, but no focus of pus could be found.

This condition remained much the same for some five months, the symptoms of myelitis becoming gradually worse until death occurred. Towards the latter part of this time, the control of the bladder and the bowels was lost, and a bed sore developed on thesac rum and right heel.

The post-mortem was made by Dr. Burr, who gave me the following notes of it and the spinal cord.

At the time of the man's death, there remained the paralysis and the contractures of the leg as before noted. There was no angularity of the spinal column and no prominence looking like an abscess near the surface. On cutting down on the spine, there was opened a large pocket holding almost a pint of pus.

This was found under and to the inner side of the right scapula. The pus had burrowed into the posterior mediastinum, but not into the pleural cavity. The laminæ of the dorsal vertebræ from the fifth down were carious. The bodies of the same vertebræ were markedly carious and filled with cheesy matter. The most marked destruction of bone was in the fifth, sixth and seventh dorsal vertebræ. The meninges of the cervical portion of the cord were normal. Those of the lumbar region were almost free of deposit. In the dorsal region, the dura was greatly thickened. At the point of most disease, from the fifth to the eighth dorsal vertebra, the cord was completely surrounded by a thickened dura, at some places one half of an inch in thickness. The brain was normal. The lungs showed miliary tuberculosis with pleural adhesions.

The microscope shows the extreme dural thickness to be due to an inflammatory process in the dura itself, with new cell formation, and to a cellular and a cheesy deposit on the outer side of the dura.

The cord is greatly pressed upon by this new tissue. It is distorted and nearly destroyed at this site by both the mechanical and some inflammatory processes. Secondary degenerative lesions are found both above and below this point. At the place of greatest pressure, there is shown some inflammatory changes. Many new cells are present where only nervous tissue should exist.

There is no distinct line of demarcation between the gray and the white matter. Both being almost totally destroyed. The cells in the anterior horns of the gray matter are few in number. Some are destroyed. Others are not perfect, having lost some of their processes. The central canal is filled by new epithelial cells. The white matter is mostly degenerated in all portions of the cord at this level. Most of the axis cylinders and the myelin sheaths are totally destroyed. Other myelin sheaths are undergoing destruction, being swollen or narrowed, according to the degree of degeneration. Small particles of myelin, not in cylindrical form, are everywhere found through the section. There are a few of both myelin sheaths and axis cylinders that are normal. These are found near the centre of the section, and around the remains of the gray matter where least pressed upon.

The secondary degeneration both above and below the point of most pressure shows the same kind of

change as above described,but to a less extent. It follows fixed columns. In none of these is the degeneration so marked as at the transverse myelitis.

In the cervical enlargement, the posterior median tracts of Goll are almost totally destroyed. The columns of Burdock are a very little affected. There is another small degenerated area in the lateral ascending columns. This is close to the margin of the cord and in the extreme lateral part.

A section very high, near the medulla shows the degeneration in the posterior median columns. That in the ascending lateral tract is not so wide as in the cervical enlargement. The other lateral tracts are normal above the lesion.

Below in the lower dorsal region all the lateral columns are involved, except the lateral fundamental zone lying near the anterior gray matter. The direct pyramidal tracts are also marked by the degeneration.

In the lumbar region, the antero-lateral tracts all show more or less destruction, except immediately around the anterior gray matter. The degeneration is greatest near the surface.

Below the lesion the posterior columns are not affected.

I wish to thank Drs. Burr and Willits for the privilege of presenting this case.

FIG. 1. Drawing showing the gross lesions: a, right third nerve adherent to fibroid mass; b, right internal carotid plugged by a thrombus; c, fibroid mass filling the Sylvian fissure and extending backward to the crus; d, chiasm; e, left internal carotid containing recently organized thrombus; f, left third nerve; g, left posterior communicant at junction with postcerebral; h, basilar giving off postcerebral. *

* This illustration is used through the courtesy of the publishers of the Medical News.

FIG. 2. Section of right oculo-motor nerve, showing complete degeneration and almost total disappearance of the nerve fibres. Low magnification.

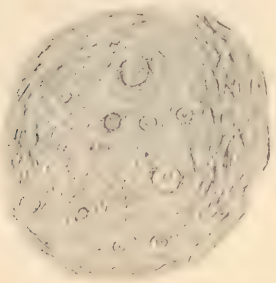

FIG. 3. High magnification of FIG. 2.

CEREBRAL SYPHILIS.[1]

By MARY ALICE SCHIVELY, M D.

THE case under examination presented the following clinical history: L. S., single, aged 29 years; a seamstress by occupation. The family history was negative. She had a history of alcoholism and syphilis, with sore throat, skin eruption and loss of hair. In July, 1894, she began having persistent frontal headache; at the same time her eyesight began to fail. Upon awakening one morning early in September, she noticed for the first time that her right eye turned upward and outward. Upon attempting to rise she was unable to stand, and discovered that her left leg was useless. She afterwards regained some use of her leg, but continued to be subject to occasional attacks, during which it would suddenly give way.

The patient was admitted to the Nervous Wards of the Philadelphia Hospital in December, 1894.

At this time she had a stupid expression of countenance, and wandered about aimlessly, speaking but little. She constantly complained of headache which was relieved somewhat at intervals, by repeated doses of potassium iodide. She had periods of marked irritability, which were followed in turn by mental hebetude, after which she would remain in bed and during which she could not easily be aroused, and would not answer questions. Her gait was of a shuffling character; knee-jerk was plus and there was slight ankle-clonus of the left side.

Examination of the eyes was made by Dr. Oliver The conditions were as follows: There was complete ptosis of the right eyelid; this eye itself was directed outward, and all movements of the globe were lost, except outward and a slight movement upward and outward; the pupil was immobile, four mm. in diameter.

[1] For a short report of this case, exclusive of the results of the microscopical examination, consult Mills "Some Phases of Syphilis of the Brain," Case V., *Medical News*, December 7, 1895.

The left pupil responded to light. Both pupils were oval and both optic discs gray.

On February 28, 1895, the patient went to bed in a spastic condition similar to that of previous attacks, but more marked. The left side of the face was flushed and swollen and she spoke only in monosyllables. During the night she had a convulsive attack followed by general muscular twitchings. After this the oculo-motor paralysis became deepened and she would not answer questions, although she appeared to understand what was said to her.

During the next day the mental dullness deepened gradually into coma. On the following day there was stiffness of the right arm, but relaxation of the left arm and both legs; K. J. was exaggerated on the left side. The left eye remained open and fixed, the pupil being contracted. Later there was Cheyne-Stokes breathing. The patient remained in an apoplectic condition, dying on the evening of the second day.

The post mortem examination revealed the following conditions: The dura was normal and not adherent; the pia free from inflammation, but much thickened; the meningeal vessels showed distinctly on the surface, the vessels over the hemisphere were distended. The right internal carotid was free until within two or three mm. of the penetration of the dura, and contained a flesh-colored, well organized thrombus. (Fig. 1). The right posterior communicating artery was a mere filament while the left was much enlarged. The right anterior cerebral was very small; the left anterior cerebral, very large. To the right side of the chiasm and the beginning of the optic nerve, there was found a dense yellow mass surrounding the internal carotid and adherent to the oculo-motor and optic nerves; this mass fairly obliterated the Sylvian fissure in which it was lying. The left internal carotid contained a recently organized thrombus in the region from which the posterior communicating and anterior and middle cerebral form; this thrombus extended into the middle cerebral artery for about one half its length. The cerebellum, pons and medulla showed on section nothing abnormal. Upon section of the right hemisphere, the head of the caudate nucleus, the anterior extremity of the striate body and the lenticular nucleus were found to be softened. The material in this softened cavity was yellow and about the appearance of pus.

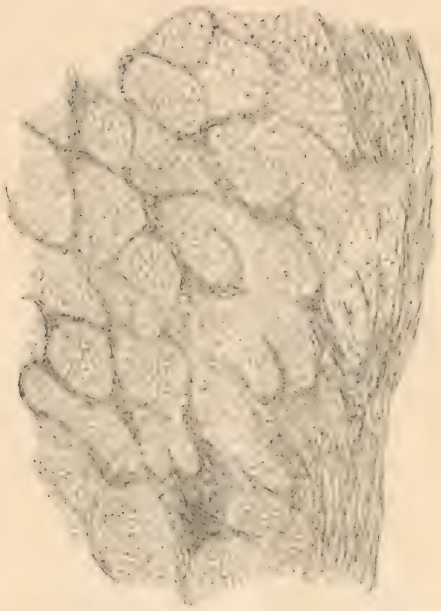

FIG. 4. Section of right optic nerve, showing new fibrous formation, round cell infiltration, and some degeneration of the nerve fibres.

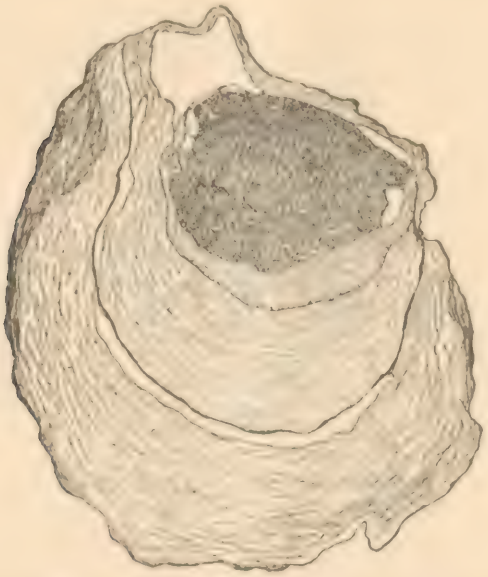

FIG. 5. Section of the left internal carotid artery, showing a recently organized thrombus, and arteritis proliferans nodosa.

FIG. 6. Section of the right internal carotid artery, showing obliterating endarteritis, thrombus formation, and subsequent caseation of the entire structure.

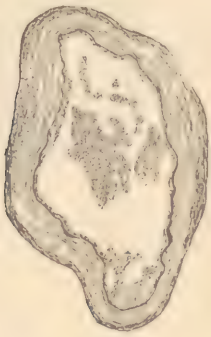

FIG. 7. Section of the right posterior communicating artery.

Through the kindness of Dr. Mills, I obtained the above notes of the case from the Philadelphia Hospital Records, and also the specimen for microscopic study. The brain was hardened in Müller's fluid, and the sections were stained with hæmatoxylin and eosin; hæmatoxylin, picric acid and fuchsin; and according to the Weigert Pal method. As a result of the microscopical examination, I offer the following facts illustrated in the accompanying drawings and photographs.

The oculo motor nerve of the left side shows in section the presence of scattered regions of small, round-celled infiltration, and some tendency to fibrous formation. The small, round-celled infiltration follows the course particularly of the endoneurium, separating the nerve tracts into smaller bundles, and that of the blood vessels. The blood vessels themselves are dilated; their walls thickened and surrounded frequently by groups of these small cells.

In one portion of the margin of the nerve nearest to the median line of the brain, there is some evidence of degeneration of the nerve fibres. Elsewhere the structure proper of the nerve is normal; the axis cylinders being distinct.

The oculo-motor nerve of the right side shows complete degeneration and almost total disappearance of the nerve fibres. (Figs 2 and 3). Only the remnants of a few degenerated nerve fibres are seen, the nerve tissue being replaced by fibrous tissue which is, in turn, in various stages of casecation. The caseous remnants of a few minute blood-vessels can be detected. The position of the groups of partially degenerated nerve fibres is central and lateral. The epineurium is infiltrated with small, spheroidal, densely staining cells, and this formation is continuous with the gummatous mass which surrounds the nerve. This gummatous structure consists partly of fibrous tissue which is very dense, and is infiltrated with small cells (similar to those seen in the epineurium), while other portions consist of more or less homogeneous substance without nuclei or cellular infiltration.

The left optic nerve exhibits changes similar to those affecting the left oculo motor. While there is present very little degeneration of the nerve fibres, the round-celled infiltration and fibrous formation are more evident than in the other nerve. The epineurium, perineurium and endoneurium, even to the finest divisions

of the latter are similarly affected. The round-celled infiltration follows also the minute arteries whose walls are thickened.

The right optic nerve (Fig. 4) shows a greater extent and a more advanced stage of changes similar to those observed in the left. Particularly on the side of the nerve nearest to the gummatous mass (which invades this region of the brain), there is an interstitial infiltration of small spheroidal cells. In places these have progressed to spindle shape and fibrous formation. In some regions, especially on the side above referred to, there is in addition marked degeneration of the nerve fibres. There are present also scattered areas of degeneration in neighboring regions, but none so distinctly marked as those above described.

The left internal carotid artery (Fig. 5) shows upon examination a recently organized thrombus, which fills up the whole lumen of the vessel. There are present evidences of arteritis proliferans nodosa, the adventitia, muscular coat and intima being much enlarged upon one side, while on the opposite side there is relative thinning of the walls which are, however, thicker than normal. The adventitia is extensively infiltrated by small spheroidal deeply staining cells, which are here and there collected into dense masses. In some places there is a tendency toward caseation; this being shown by an indistinct staining of the nuclei; but the general tendency is not in this direction. The elastic lamina is intact. Evidences of obliterating endarteritis are shown by the increased thickness of the intima due to increase in size and number of the endothelial cells in addition to the spheroidal cells.

In the right internal carotid artery (Fig. 6) are seen the conditions of obliterating endarteritis, thrombus formation and subsequent caseation of the entire structure. The vessel-walls and the tissues in their immediate neighborhood are caseous for a considerable distance. The distinction of vessel-walls into adventitia, media and intima cannot be recognized, and there is no elastic lamina visible. In the periphery of the adventitia there is an invasion of small, deeply staining spheroidal cells on that side of the artery, which is nearest to the surrounding gummatous mass. This gummatous growth filling up the Sylvian fissure extends about half-way around the artery, and consists peripherally of dense granulation tissue; of small round cells with more cen-

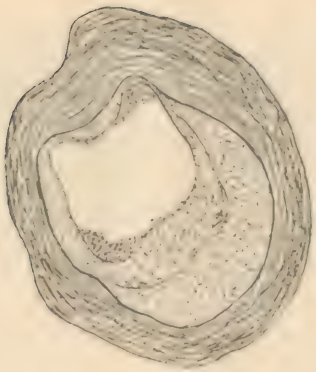

FIG. 8. Section of the left posterior communicating artery.

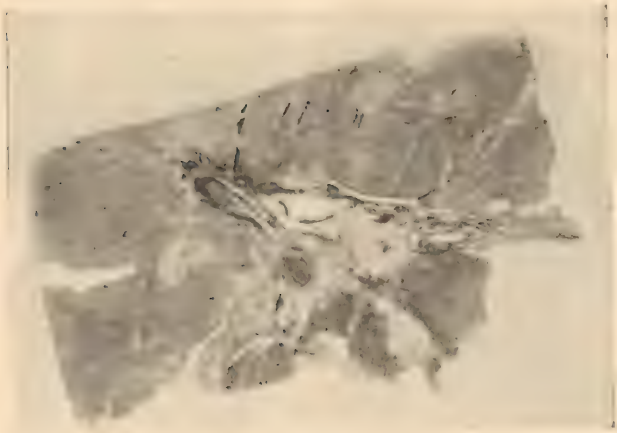

FIG. 9. Section through the right Sylvian fissure, showing gummatous formation and caseati n, endarteritis and periarteritis, and involvement of the meninges and brain substance.

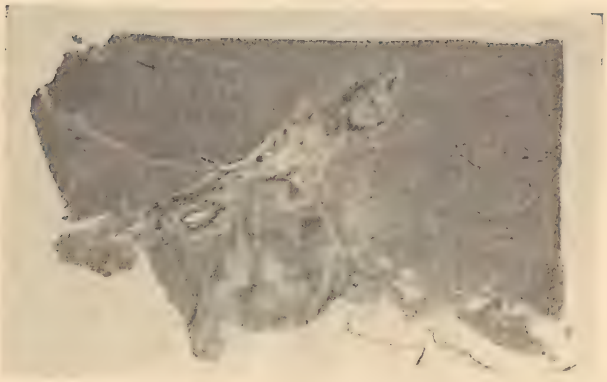

FIG. 10. Section through the right Sylvian fissure, showing changes similar to those in Fig. IX.

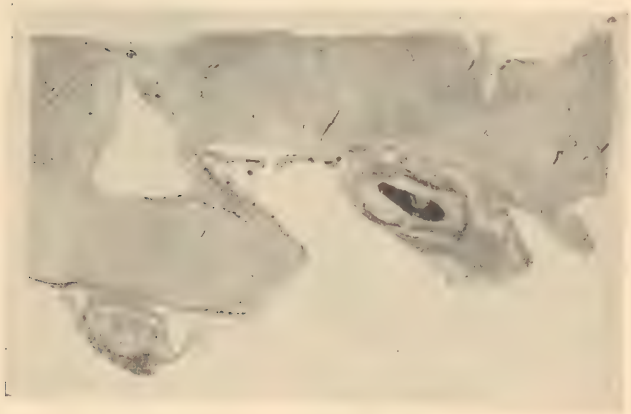

FIG. 11. Section through the left Sylvian fissure, left carotid artery, and optic chiasm, showing thrombus in left carotid artery and minor grades of other changes seen in FIGS. IX and X.

trally located fibrous tissue, infiltrated here and there with spindle and round cells; the centre being a great mass of caseated tissue. The small cells are arranged in definite areas in some portions, but for the most part irregularly scattered.

The right (Fig. 7) and left (Fig. 8) posterior communicating arteries exhibit earlier stages, and minor degrees of changes similar to those affecting the carotid arteries.

A section through the right Sylvian fissure and the gumma (Figs 9 and 10) of that region, scarcely permits of a distinction between brain cortex, meninges and vessels. Obliterating endarteritis and periarteritis have progressed in this region until the vessel and its thrombus form the centre of a caseous gummatous mass, around which are regions of fibrous and granulation tissue. There are numerous other foci of caseation of lesser extent alike surrounded by fibrous tissue, spindle and small round celled infiltration. The portion formerly meninges, is now a layer of densely packed small round cells, and partly spindle celled and fibrous or caseous. The infiltration extends a varying distance into the cerebral cortex in places, there being no area of limitation between cortex and meninges. The blood-vessels of the cortex are dilated the walls thickened and surrounded by the same small, round celled infiltration.

A section through the left internal carotid and the optic chiasm (Fig. 11) reveals changes similar to, although less marked than those present upon the opposite side. The chiasm exhibits the already described disease of the support structures and some disease of the nerve elements. The implication of the blood vessels is marked.

The pons Varolii shows evidences of gummatous formation, with caseation in the region of the pyramidal tract of the right side. The vessels of the pia mater in this region are enlarged and surrounded by spheroidal cells; the walls of these vessels are much thickened. There is some degeneration of the fibres of the pyramidal tract of the right side. In other respects the pons is normal.

In conclusion, the leading clinical features of the case may be summarized as follows: right oculo-motor paralysis; paresis of the left leg; mental symptoms (aphasia, irritability, stupor) and a long apoplectiform period with convulsions preceding death. Pathologically, the case illustrates most beautifully the multiplicity and divers

ity, both in nature and distribution, of the lesions of cerebral syphilis. The multiplicity of the lesions is evident in the various involvements of the blood-vessels, meninges, nerves and brain substance. The diversity in the nature of the lesions is manifest, when we review in detail the arteritis proliferans nodosa, or again the arteritis obliterans simplex leading in places to multiple thromboses and localized softenings; the interstitial neuritis with degeneration of the nerve fibres, or the absolute syphilitic destruction of the nerve; the meningeal implication progressing in places to simple round-cell infiltration or fibrous transformation and fusion with the brain substance, in others to caseation; and the involvement of the minute vessels of the brain, which tissue is itself in places slightly implicated, again extensively so, as evidenced by softening, or loss of normal limitations and involvement in large gummatous formations, as in the pons and Sylvian fissure. There is hardly a manifestation of syphilis, possible in the structures examined, that does not here find its exemplification.

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